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Sequential activity in asymmetrically coupled winner-take-all circuits

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Sequential Activity in Asymmetrically Coupled Winner-Take-All Circuits

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Understanding the sequence generation and learning mechanisms used by recurrent neural networks in the nervous system is an important problem that has been studied extensively. However, most of the models proposed in the literature are either not compatible with neuroanatomy and neurophysiology experimental findings, or are not robust to noise and rely on fine tuning of the parameters. In this work, we propose a novel model of sequence learning and generation that is based on the interactions among multiple asymmetrically coupled winner-take-all (WTA) circuits. The network architecture is consistent with mammalian cortical connectivity data and uses realistic neuronal and synaptic dynamics that give rise to noise-robust patterns of sequential activity. The novel aspect of the network we propose lies in its ability to produce robust patterns of sequential activity that can be halted, resumed, and readily modulated by external input, and in its ability to make use of realistic plastic synapses to learn and reproduce the arbitrary input-imposed sequential patterns. Sequential activity takes the form of a single activity bump that stably propagates through multiple WTA circuits along one of a number of possible paths. Because the network can be configured to either generate spontaneous sequences or wait for external inputs to trigger a transition in the sequence, it provides the basis for creating state-dependent perception-action loops. We first analyze a rate-based approximation of the proposed spiking network to highlight the relevant features of the network dynamics and then show numerical simulation results with spiking neurons, realistic conductance-based synapses, and spike-timing dependent plasticity (STDP) rules to validate the rate-based model.

1 Introduction ---

The ability to recognize sequences of sensory stimuli and respond using a set of coordinated and sequential motor activities is one of the hallmarks of

intelligent behavior (Lashley, 1951). Underlying this ability in many cases is neural activity that unfolds sequentially in time, during both perception (Dehaene, Changeux, & Nadal, 1987; Pulvermüller & Shtyrov, 2008) and the production of sequential motor activity (Tanji, 2001; Hahnloser, Kozhevnikov, & Fee, 2002). This neural activity typically represents transient or periodic responses of recurrent neural networks, which take the form of a sequential activation of a number of neurons or neuron pools. Computational models of networks that exhibit sequential patterns of activity typically make use of asymmetric connections that guide activity from one neuron pool to the next. For example, asymmetric feedforward excitatory connections are a prominent feature in synfire chain models (Abeles, 1982), while asymmetric inhibitory connections have been shown to play an important role in metastable networks of competing neurons used to generate patterns of sequential activity (Rabinovich et al., 2001; Afraimovich, Zhigulin, & Rabinovich, 2004; Rabinovich, Huerta, Varona, & Afraimovich, 2008). In unstructured networks that do not have explicit asymmetric connectivity patterns, short- or long-term plasticity mechanisms can lead to the formation of asymmetric connections, which can then give rise to sequential patterns of activity (e.g., see Fiete, Senn, Wang, & Hahnloser, 2010). Competitive recurrent network models can use different types of adaptation mechanisms to produce sequential patterns of activity (Hopfield, 2010; Deco & Rolls, 2005; Verduzco-Flores, Bodner, & Ermentrout, 2012). The adaptation mechanisms present in these networks reduce the activity of the active group of neurons in the network so that a different set of (nonadapted) neurons can win the competition and suppress the adapted set of neurons (which then can slowly recover from the adaptation). To generate patterns of sequential activity that can be halted and resumed by external input, a network requires a structure of stable fixed points of activity, or attractors, which can store the current state of the sequence. Sequential activity in attractor networks like the Hopfield network (Hopfield, 1982) can be obtained by introducing asymmetric connections that guide the activity from one attractor to the next (Amit, 1988; Kleinfeld & Sompolinsky, 1988). During spontaneous sequential activity, the asymmetric connections should be slow or exhibit a delay that is long enough to allow the fast symmetric connections to stabilize one attractor before the asymmetric connections induce a transition to the next attractor. If the asymmetric connections are weak enough, the transition between attractors can be triggered and controlled by the application of external input signals. The Hopfield network dynamics, however, are highly simplified, and it is questionable whether the phenomena arising from these dynamics can be reproduced in biologically realistic network models.

In this letter, we present a recurrent network model that makes use of asymmetric connections to induce transitions between attractors but does not require the artificial separation between slow and fast synapses or the fine-tuning of synaptic weights. The network we propose has a cortically

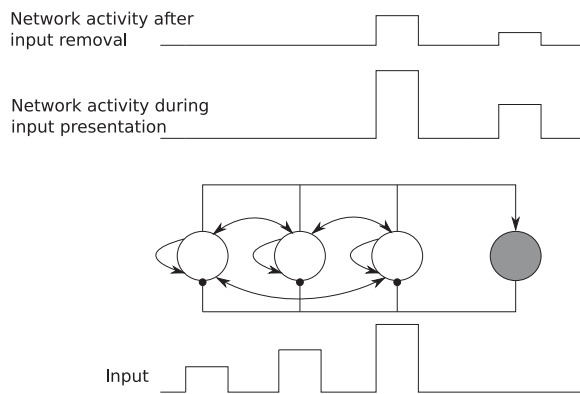


Figure 1: Schematic diagram of a winner-take-all network. Excitatory populations are shown as white circles, inhibitory populations as gray circles. The network selectively amplifies the strongest external input, suppressing the weaker ones. After the external input is removed, the network settles into the attractor state, which is characterized by persistent activity in the excitatory population that received the strongest input.

inspired architecture with realistic neuronal and synaptic dynamics; it exhibits stable transitions from one state to another and can learn to reproduce temporal sequences imposed by the input signals by making use of plastic feedforward excitatory synapses. The basic building block of the proposed network is a winner-take-all (WTA) network of the type shown in Figure 1. These types of networks can have multiple attractor states, characterized by heightened activity in a single excitatory population that persists after external input is removed. The specific attractor state chosen is a function of the inputs received by the different excitatory populations. The network imposes an interpretation on the noisy or incomplete input by displaying one of a number of stereotypical responses. The connectivity patterns of these WTA networks are consistent with both intracellular recordings in cat visual cortex (Douglas, Martin, & Whitteridge, 1989; Douglas & Martin, 1991) and anatomical connectivity data measured in cat visual cortex and rat auditory cortex (da Costa & Martin, 2010; Martin, 2011), which point to a high level of recurrency with many excitatory, inhibitory, and excitatory-inhibitory loops (Binzegger, Douglas, & Martin, 2004; Thomson, West, Wang, & Bannister, 2002). These excitatory-inhibitory recurrent loops can produce oscillatory patterns of activity if the time constant of the inhibitory population in the WTA network is made sufficiently long compared to the time constant of the excitatory populations (Ermentrout, 1992). In the presence of asymmetric connections between the excitatory populations within the WTA network, these oscillatory dynamics can facilitate the transition of activity from one excitatory population to the next by restarting

the winner selection process at the peak of the inhibition. However, the requirement for slow inhibitory neurons is at odds with the observed high excitability of GABAergic interneurons (McCormick, Connors, Lighthall, & Prince, 1985) of which many classes make synapses on the soma, axon, or proximal dendrites of pyramidal cells (Freund, Martin, Smith, & Somogyi, 1983; Markram et al., 2004) leading to fast and strong inhibition. It has been argued that the WTA networks of the type depicted in Figure 1 represent a potential cortical circuit motif, present across the cortices of many species (Douglas & Martin, 2004).

The recurrent network proposed in this work is composed of multiple WTA circuits, connected to each other by a coupling scheme based on the interlayer asymmetric connections observed in cat and rat neocortices (Watts & Thomson, 2005). The parameters for each WTA circuit are chosen to ensure the existence of stable attractor states in the WTA circuit when it is uncoupled from the rest of the network. These attractor states provide a mechanism that restores the network activity to a well-defined level at each step of the sequence. This signal restoration mechanism is crucial to prevent small amounts of noise in the system from extinguishing the sequential activity. Anatomically realistic asymmetric connections between the WTA circuits elicit transitions between the different WTA attractor states. These asymmetrically coupled WTA circuits are thus able to sequentially visit a number of well-defined states without settling into one of them and bringing the sequence to a halt. The sequence of activity produced by the network is robust to noise with little risk of the activity dying out or being driven into ill-defined states by small perturbations. The network supports the propagation of sequential activity along a number of possible paths. The path actually chosen depends on the relative strengths of the asymmetric coupling connections. If these connections are weakened, transitions between attractors are no longer spontaneous but depend on external input to trigger them. External input can also influence the path the sequential activity takes.

The dynamics of different variants of WTA circuits have been extensively analyzed using rate-based models (Rutishauser, Slotine, & Douglas, 2012). Different WTA coupling schemes have also been proposed that can elicit spontaneous or triggered transitions between different attractor states (Rutishauser, Douglas, & Slotine, 2011; Rutishauser & Douglas, 2009). In this letter, we extend these studies by considering both rate-based and spiking models of coupled WTA circuits that exhibit realistic dynamics. We introduce a novel and anatomically justified scheme for coupling the WTA circuits. We also investigate the role of learning and show how plastic synapses enable the network to learn to reproduce input-imposed sequences.

In the next section, we describe and analyze the rate-based version of the proposed network, where activity in each neural population is represented by a single dynamical variable, and show how stable sequences can be generated. We introduce synaptic plasticity into the asymmetric connections

and show how the network can learn to reproduce sequences imposed by external input. In section 3 we describe the spike-based implementation of the network. The spiking network is composed of integrate-and-fire neurons that communicate using conductance-based synapses. Learning is done through a spike-based plasticity rule that modulates the strength of the feedforward synapses. In section 4 we discuss the results obtained and present our conclusions in section 5.

2 Network Description

2.1 Network Architecture. Figure 2 shows the full connectivity profile of the proposed network. The diagram shows a network composed of three coupled stages. Each stage is a WTA circuit whose parameters are configured so that it has nonzero stable attractor states in the absence of input when it is uncoupled from the rest of the network. Feedforward excitatory connections connect the excitatory populations of one stage to the excitatory populations of the next stage in an all-to-all fashion. The excitatory populations of one stage project back to the inhibitory population of the previous stage. There is no limitation on the number of stages that can be connected sequentially in this manner.

There is evidence that many cortical interlayer connections are selective and asymmetric, that is, nonreciprocated (Thomson, Bannister, Mercer, & Morris, 2002). Reconstructions of neural circuitry in the cat visual cortex point to a major loop made of nonreciprocated pyramidal-pyramidal connections (Binzegger et al., 2004). The glutamergic connections forming the loop proceed from layer 4 to layer 2/3 to layer 5 to layer 6 and back to layer 4. While the feedforward projections from layer 4 to layer 2/3 and from layer 2/3 to layer 5 target pyramidal cells and, to a lesser degree, interneurons, there exist feedback projections from layer 5 to layer 3 and from layer 3 to layer 4 that mainly target interneurons (Watts & Thomson, 2005). This is consistent with the connectivity profile of our network, which is characterized by nonreciprocated feedforward excitatory-excitatory and feedback excitatory-inhibitory connections. This assumes that the individual WTA networks are largely localized within individual layers, an assumption that is partly justified by dense intralayer recurrent excitatory connections and the tendency of inhibitory interneurons to arborize locally within a single layer (Douglas & Martin, 2004).

The behavior of the network in Figure 2 can be qualitatively understood by considering a single WTA stage in isolation. The excitatory populations in a WTA stage are driven by an external input or recurrent excitation, or both. If some or all of the neurons in one of the excitatory populations are receiving a total input current that is larger than the current dissipated by various leak mechanisms, these neurons will become active. Due to the recurrent excitatory connections, the firing of a subset of neurons in an excitatory population will provide additional current to other neurons in

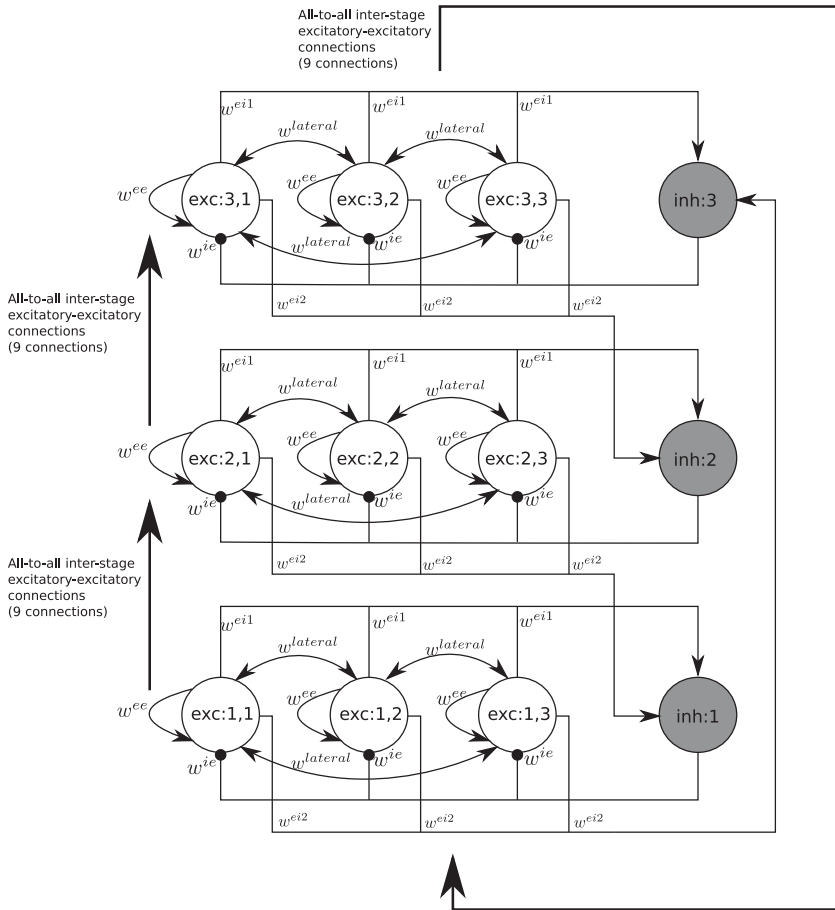


Figure 2: Three asymmetrically coupled WTA stages connected in a loop. Excitatory populations are shown as white circles, inhibitory populations as gray circles.

the population, which will cause some of these neurons to increase their activity. Eventually the average firing rate in the population will reach a point where the synaptic current coming through the recurrent connections is large enough to maintain a self-sustaining rate that persists even after all external inputs are removed. Beyond the self-sustaining rate, the average firing rate in the population will ramp up quickly until it is large enough to trigger activity in the inhibitory population through the relatively weak excitatory-to-inhibitory synapses. Activity in the inhibitory population will begin curbing the runaway excitation in the winning excitatory population.

Activity in the winning excitatory population will keep on ramping up, but at a slower rate. Activity in the inhibitory population will ramp up as well to match the increasing excitation. The increasing level of inhibition will shut down excitatory populations whose average firing rate is lower than that of the winning population. In an isolated, uncoupled WTA stage, the recurrent excitation in the winning population will eventually reach a stable equilibrium with the inhibition and the excitatory population will settle into a steady-state firing rate. At this condition, the network has settled into a stable attractor.

If the WTA circuit is asymmetrically coupled to other WTA circuits, as in Figure 2, and if the connection weights are appropriately chosen, the WTA stages will not be able to settle into stable attractors: the WTA will no longer have a constant nonzero persistent pattern of activity in the absence of external input. For example, as soon as activity in the bottom WTA circuit of Figure 2 becomes sufficiently high, the middle WTA circuit is activated and, through the feedback excitatory-inhibitory connections, it raises the level of inhibition in the bottom WTA circuit, effectively killing the activity there.

2.2 Rate-Based Implementation. To quantitatively analyze the network shown in Figure 2, we abstract the neural activity in each population and represent it by a single dynamical variable. This dynamical variable represents the average firing rate of the neurons in the population. Let R be the number of stages or WTA circuits and C the number of excitatory populations in each WTA circuit ($R = 3$ and $C = 3$ in Figure 2), and let the R stages be connected in a loop as in Figure 2; then the full firing rate model is given by

$$\begin{aligned} \tau_e \dot{x}_{j,k}^{exc}(t) + x_{j,k}^{exc}(t) = & \left[w^{ee} x_{j,k}^{exc}(t) - w^{ie} x_j^{inh}(t) + \sum_{\substack{l=1 \\ l \neq k}}^C w^{lateral} x_{j,l}^{exc}(t) \right. \\ & \left. + \sum_{l=1}^C w_{j-1,l,k}^f x_{j-1,l}^{exc}(t) - T_e + I_{j,k}^{exc} \right]^+ \\ & j = 1, \dots, R \quad k = 1, \dots, C, \end{aligned} \quad (2.1a)$$

$$\begin{aligned} \tau_i \dot{x}_j^{inh}(t) + x_j^{inh}(t) = & \left[\sum_{l=1}^C w^{ei1} x_{j,l}^{exc}(t) + \sum_{l=1}^C w^{ei2} x_{j+1,l}^{exc}(t) - T_i \right]^+, \\ & j = 1, \dots, R \end{aligned} \quad (2.1b)$$

where τ_e and τ_i represent the excitatory and inhibitory population time constants, $[x]^+ = \max(0, x)$, and all the additions and subtractions in the

subscripts wrap around to stay in the range $[1, R]$. The activity in the k th excitatory population in stage j is $x_{j,k}^{exc}(t)$, and the external input to that population is $I_{j,k}^{exc}$. x_j^{inh} is the activity of the inhibitory population in stage j . w^{ie} , w^{ei1} , w^{ei2} , w^{ee} , and $w^{lateral}$ are fixed weights (shown schematically in Figure 2). $w_{m,n,p}^f$ is the fixed feedforward connection weight connecting population n in stage m to population p in stage $m+1$ where the last addition wraps around in the range $[1, R]$. T_e and T_i are the thresholds for activating the excitatory and inhibitory populations, respectively. If the total input to a population is clamped at x^{fixed} , then the steady-state activity of this population is a linear threshold function $[x^{fixed} - T]^+$ of x^{fixed} where T , the threshold, is a population parameter. A linear threshold activation function is a good approximation of the steady-state average firing rate in a population of constant leak integrate-and-fire neurons receiving noisy, uncorrelated inputs (Fusi & Mattia, 1999). In principle, for a step increase in mean input, the actual average firing rate in a population of neurons settles into a steady state after a number of transient modes have died out (Mattia & Del Giudice, 2002; Knight, 2000), but in equation 2.1, we assume the firing rate approaches steady state only through first-order dynamics.

Note that the linear threshold activation function is nonsaturating, so any nonzero stable steady state that the network might have must be due to the interaction between the excitatory and inhibitory populations. Appendix A contains an approximate analysis of the network shown in Figure 2 and described by the model in equation 2.1.

Figure 3 shows numerical simulation results when a sequence is launched by exciting the bottom WTA stage. The simulation uses the Euler method with a time step of 0.1 ms. The weights of the feedforward excitatory-excitatory connections were independently sampled from a uniform distribution. The population parameters and connection weights are given in Table 1 of appendix A. The asymmetric connections destroy the fixed-point attractors in each WTA stage but do not completely distort the phase space around these destroyed attractors. The regions around the destroyed attractors still exert a pull on nearby trajectories, and the trajectory speed in these regions is small. These are remnants of the dynamical features of the destroyed attractor. We call these regions the “ghosts” of the destroyed attractors.

Although there can be no sustained activity state in which one of the WTA stages is individually active, the ghosts of the destroyed attractors in each stage still restore the activity toward a well-defined level. The path taken by the sequential activity is a function of the feedforward excitatory-excitatory connections linking the consecutive stages. When an excitatory population has almost “won” in stage j and is approaching the ghost of the destroyed attractor, its heightened activity is sufficient to activate the excitatory populations in stage $j+1$. Since only one winner can emerge, only the population in stage $j+1$ receiving the strongest projection weight

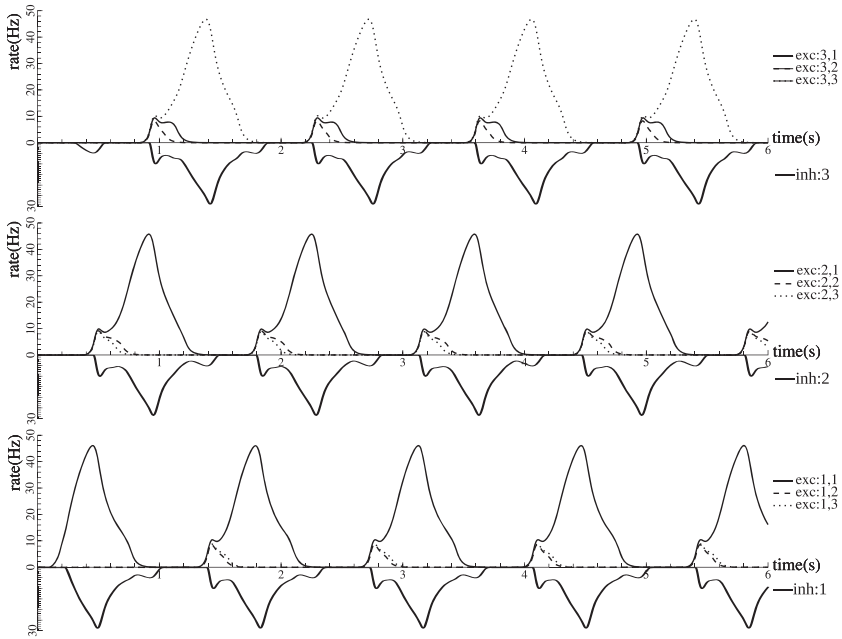


Figure 3: Simulation results showing the activity in each population of the network of Figure 2. The activities of inhibitory populations are plotted on reversed y -axes. A brief excitatory input is delivered to the exc:1,1 population at 0.1 s to launch the sequence.

from the active population in stage j will approach the ghost of the destroyed attractor and win the competition, and in the process kill the activity in stage j through the feedback excitatory-inhibitory connection. In other words, activity will jump from population exc: j,k to population exc: $j+1,p$ only if

$$w_{j,k,p}^f > w_{j,k,h}^f \quad \forall h \in \{1, \dots, C\} - \{p\}. \quad (2.2)$$

Since we connect the last (top) stage to the first (bottom) stage, the sequential activity will persist indefinitely. However, if we break the connection between the last and the first stage, the stable attractor states of the last WTA stage are restored, so that when activated, the last stage shuts down the previous stage and settles into its stable state of persistent activity. It is also possible to choose the population parameters and the intrastage connection weights in the last stage so that activity ramps up in the last stage to a level that is sufficient to shut down the previous stage. Once the previous stage is shut down, activity decays back to zero without activating subsequent stages.

2.3 Noise Sensitivity. The active restoration of sequential activity at each step adds significant robustness to noise. Each step of the sequence is well defined. Activity cannot propagate along two paths at once because each WTA can support only one winner, and the winning population is always unambiguous. Furthermore, only by producing an unambiguous winner can a WTA stage activate the next stage and allow the sequence to proceed. Figure 4 shows simulation results of the network shown in Figure 2 at different noise levels. The populations were perturbed by uncorrelated white gaussian noise. Integration was done using the Euler-Maruyama method with a time step of 0.1 ms. When activity is ramping up in a WTA stage, noise can bias the competition and allow an arbitrary population to win. The amount of noise needed to override the pattern of sequential activity encoded in the feedforward excitatory-excitatory weights increases substantially if these weights are well separated. In Figure 4a, the noise level is not high enough to perturb the sequence. In Figure 4b, the high noise level perturbs the path of the sequential activity but is unable to extinguish the activity, and winner selection in each stage is still unambiguous.

2.4 Triggered Transitions. If we sufficiently reduce the strength of the feedforward interstage excitatory-excitatory connections, we are able to restore the attractor states of the individual WTA stages. When an excitatory population in a WTA stage wins the competition, it is able to settle into an attractor state without triggering the next WTA stage. It will provide a priming input only to the next stage. An external diffuse background excitation will provide the necessary additional stimulation needed to activate the primed WTA stage and resume the sequence. Removal of this input will pause the sequence in its current state. This behavior is illustrated in Figure 5, where an external diffuse input is used to halt and resume the sequence. The attractors that materialize when the sequence is halted allow the network to effectively store the current state of the sequence (i.e., the stage at which the sequence has halted and the winning population of that stage).

2.5 Sequence Learning. If activity in a network of asymmetrically coupled WTA circuits like the one shown in Figure 2 is left to spontaneously develop, the network will eventually settle into a repeating sequential pattern. The sequential pattern is composed of a number of discrete transitions where each transition is characterized by the activity jumping from one WTA stage to the next. Each of the transitions forming this repeating pattern must obey the condition given by equation 2.2. Selective external input that targets some of the excitatory populations can dictate the winning population in each WTA circuit and override the sequence encoded in the feedforward weights. After the input is removed, the externally imposed sequential pattern may persist if its constituent transitions satisfy equation 2.2. But in general, this will not be the case. This is illustrated in Figure 6.

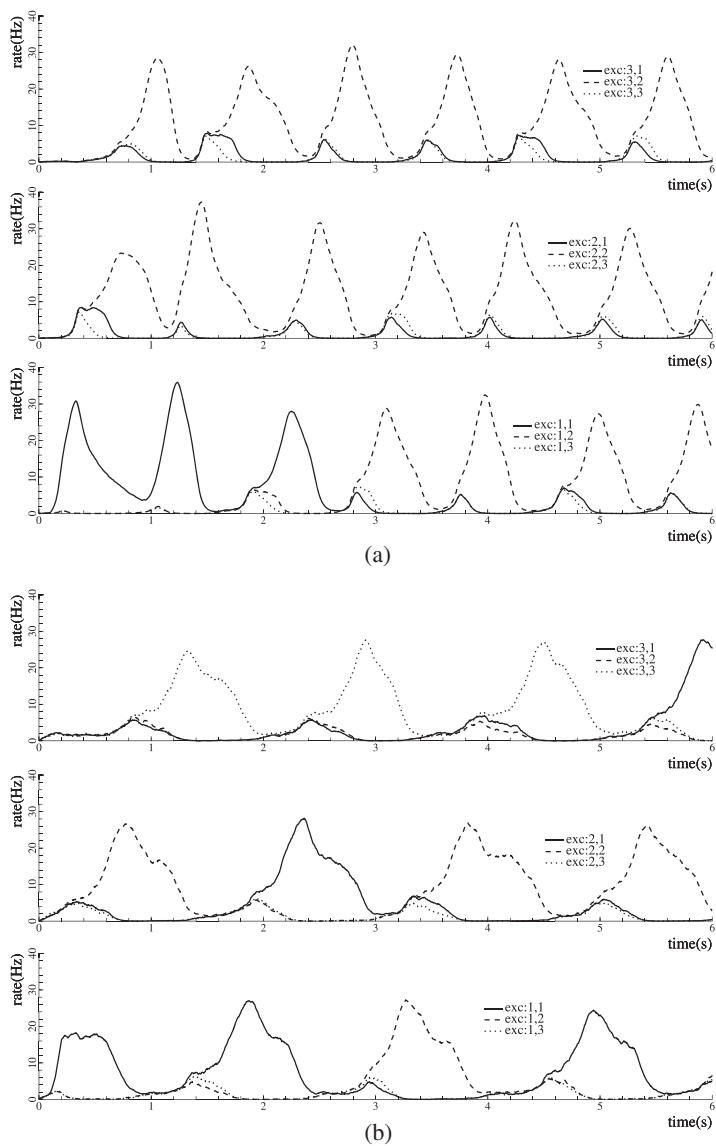


Figure 4: Activity in the excitatory populations of the network of Figure 2 when all populations (including the inhibitory populations) are perturbed by a white gaussian noise process. (a) Noise root mean square amplitude (rms) is 300. (b) Noise rms is 600.

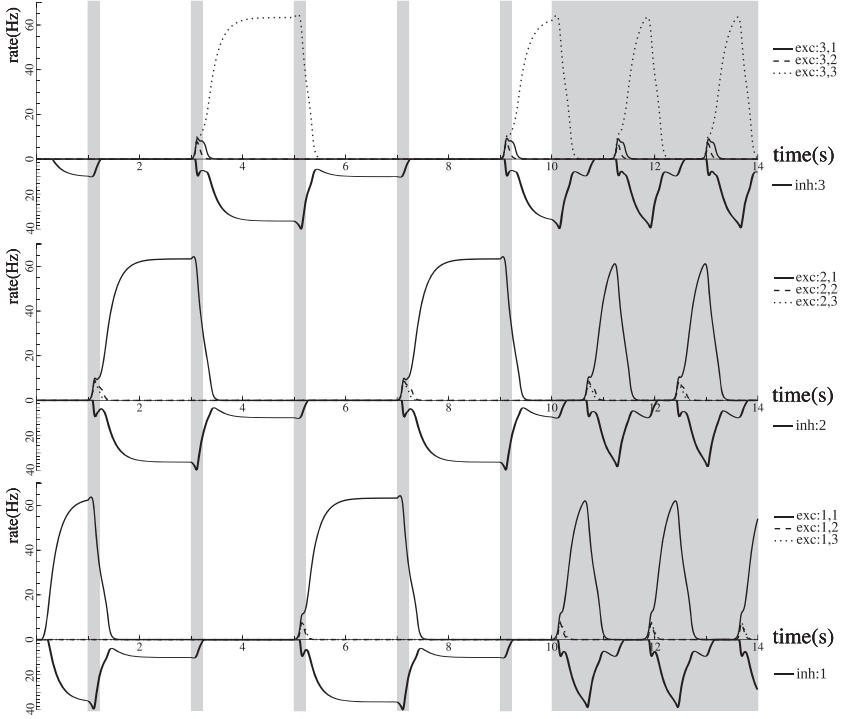


Figure 5: External background excitation was applied to all excitatory populations in the network of Figure 2 for 0.2 s at 2 s intervals starting from 1 s, and then applied continuously starting from 10 s. The plot shows the activity of excitatory populations in the network. The activities of inhibitory populations are plotted on reversed y -axes. The application times of background excitation are shown as gray rectangles in the plot. When briefly applied, background excitation triggers a single transition in the sequence; when continuously applied, the network continuously transitions through the different states.

After the external input is removed, the sequential activity is again controlled by the feedforward excitatory weights and the final pattern is different from both the initial and the input-imposed sequences.

We introduce a simple rate-based plasticity rule that operates on the feedforward excitatory-excitatory connections. The rule is given by

$$\begin{aligned} \tau_w \dot{w}(t) = & Ku(t)([v(t) - v_{th}]^+(w_{\max} - w(t)) \\ & + [v(t) - v_{th}]^-(w(t) - w_{\min})) \end{aligned} \quad (2.3)$$

$$[x]^+ = \max(0, x), \quad [x]^- = \min(0, x),$$

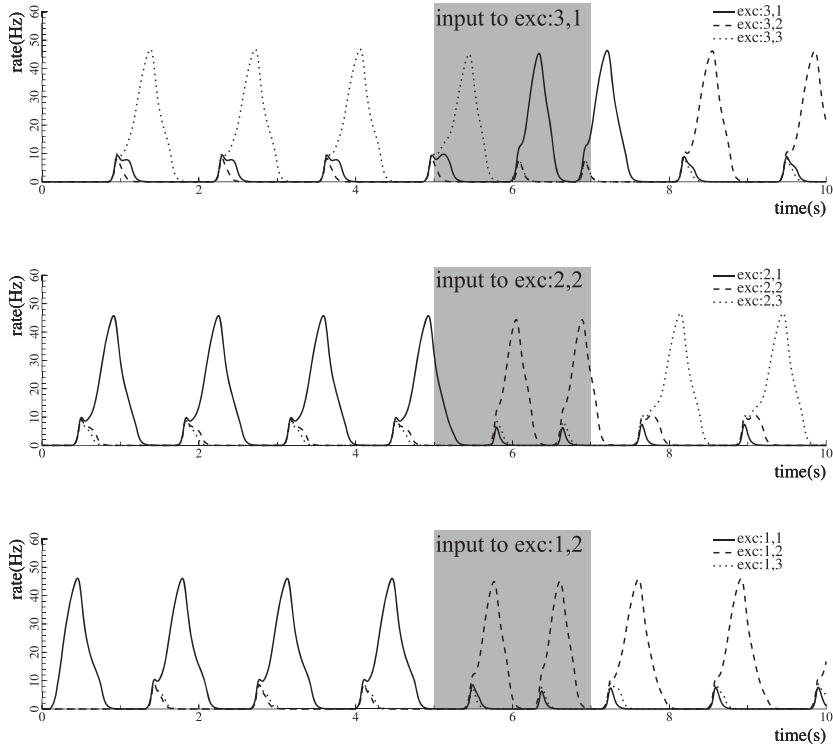


Figure 6: External input steering the path of the sequential activity. External excitation was applied between 5 s and 7 s to the populations exc:1,2, exc:2,2, and exc:3,1. External excitation application time is shown as gray rectangles on the plot. External excitation succeeds in steering the sequence, but as soon as it is removed, the input-imposed pattern is lost.

where $w(t)$ is the connection weight, $u(t)$ the source population activity, and $v(t)$ the target population activity. K is a constant that controls the learning speed, and τ_w is the learning time constant. This Hebbian plasticity rule is similar to the Bienenstock-Cooper-Munro (BCM) rule (Bienenstock, Cooper, & Munro, 1982). The main difference is that the threshold v_{th} that delimits the transition between potentiation and depression is a fixed constant. The sliding threshold used in the BCM rule is needed to keep the rule stable and avoid runaway potentiation of the weights. The plasticity rule given by equation 3.3 is trivially stable as the weight $w(t)$ is softly bound by w_{min} and w_{max} . The learning rule captures the dependence of potentiation and depression induction on the postsynaptic firing rate (Sjöström, Turrigiano, & Nelson, 2001).

When an external input imposes a particular sequence on the network, this will lead to a reorganization of the weights of the feedforward

excitatory-excitatory connections. The feedforward connections that are part of the input-imposed sequence (i.e., the connections that connect the winning excitatory population of one stage to the winning excitatory population of the subsequent stage) will potentiate. All connections going from the winning excitatory population of one stage to the losing excitatory populations of the next stage will depress. This is because activity in each excitatory population will go above v_{th} only if it is the winning population. This is illustrated in Figure 7. The K parameter in equation 2.3 was chosen so that a sequence has to propagate through the network only once to fully reorganize the feedforward connection weights in a manner that favors the regeneration of the same sequence. By choosing a smaller value for K , we can reduce the rate at which the weights change and multiple iterations of the same sequence will then be needed to fully reorganize the weights. In Figure 7, an initial external input reorganizes the plastic weights in order to store the sequence exc1,1 exc2,1 exc3,3. Later, another input imposes the sequence exc1,2 exc2,2 exc3,1 on the network and in the process reorganizes the plastic weights in order to store the imposed sequence. The input-imposed sequence persists after input removal.

3 Description of the Spiking Model

We implemented the network shown in Figure 2 using populations of spiking integrate-and-fire neurons with conductance-based synapses. Each neuron is modeled as

$$\begin{aligned} C_m \dot{V}_m(t) &= g_l(V_L - V_m(t)) + I_{AMPA} + I_{NMDA} + I_{GABA} \\ V_m(t) &\leftarrow V_L \quad \text{if } V_m(t) > V_{firing}. \end{aligned} \quad (3.1)$$

$V_m(t)$ is the membrane potential and C_m the membrane capacitance. g_l is the leak conductance and V_L the resting potential. I_{AMPA} , I_{NMDA} , and I_{GABA} are the synaptic currents due to the activation of the AMPA, NMDA, and GABA_A receptors, respectively. The neuron fires when $V_m(t)$ crosses V_{firing} . $V_m(t)$ is then reset to V_L . After firing, the neuron enters a refractory period that lasts for T_{ref} . During the refractory period, the neuron is not integrating any synaptic inputs.

We represent each excitatory population and each inhibitory population in the network shown in Figure 2 by 30 excitatory neurons and 30 inhibitory neurons, respectively. A connection between two populations is implemented by having each neuron in the source population form synapses on each neuron in the target population. The strength of each individual synapse is drawn from a random distribution. Appendix B contains a full description of the synaptic currents and the neuron and synapse parameters used in the following simulations.

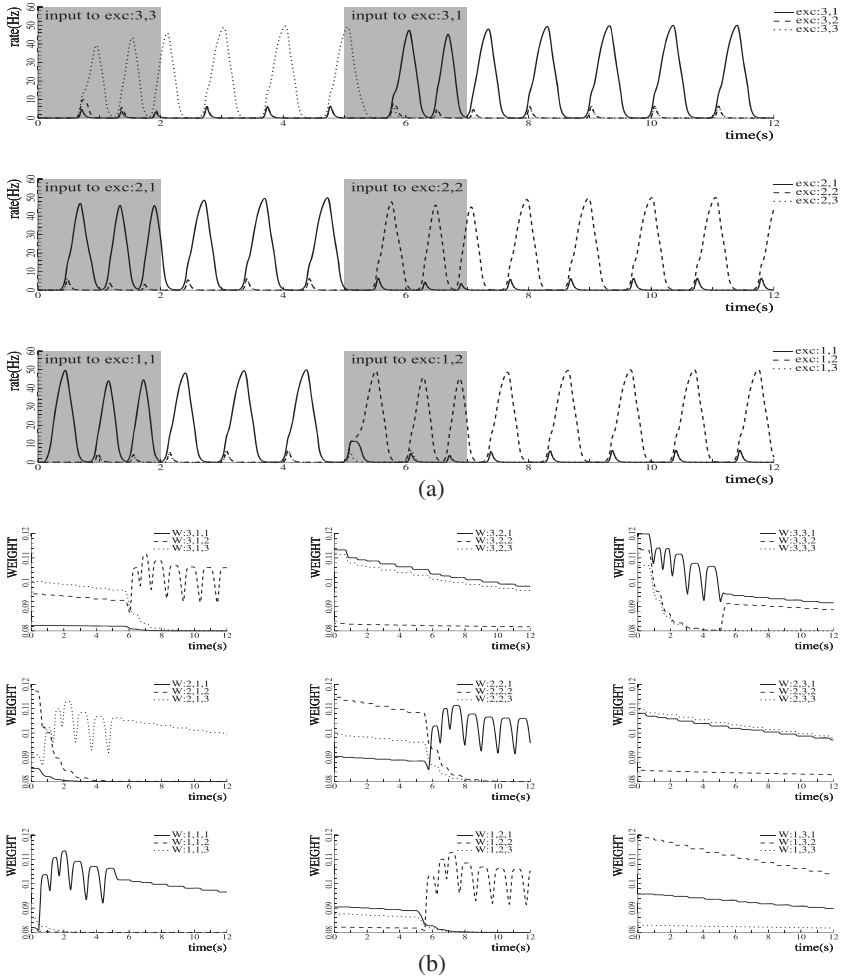


Figure 7: External input reorganizes the plastic feedforward excitatory weights to store the imposed sequence. (a) Activity of the excitatory populations in the network. Gray rectangles indicate external excitation to specific populations. (b) The strengths of the feedforward excitatory connections. $W:i,j,k$ is the connection weight from population $\text{exc}:i,j$ to population $\text{exc}:i+1,k$. The addition wraps around to stay in the range $[1,3]$.

Figure 8 shows the simulation results of the spiking implementation of the network shown in Figure 2. Sequential activity was launched by providing external excitation to the population $\text{exc}:1,1$ during the initial 0.1 s. The effect of small differences in the gaussian distributed strengths of the

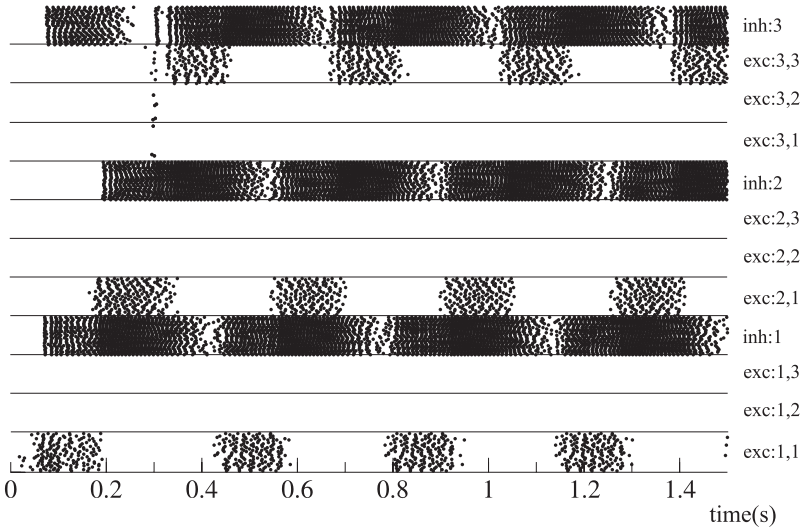


Figure 8: Spontaneous sequential activity in the network of Figure 2 using spiking neurons. Each row is a raster plot of the activity of the 30 neurons making up a population.

feedforward excitatory-excitatory synapses is amplified by the WTA mechanism. These small differences dictate the path of the sequential activity.

In Figure 9, the interstage excitatory-excitatory connections have been weakened so that activity cannot spontaneously propagate in the network. Uniform background excitation takes the form of a 300 Hz Poisson spike train that activates *AMPA*-mediated conductances in all excitatory neurons. If background excitation is briefly applied, it triggers a single transition in the sequence. If it is continuously applied, sequential activity proceeds as in the spontaneous case shown in Figure 8. As in the firing rate model, background excitation can be used to halt or resume the sequence. The sequential activity path is determined by differences in the feedforward excitatory weights that were randomly chosen at the beginning of the simulation and by the fluctuations in neural activity. The effect of the latter can be seen when the population *exc:1,3* is active. Due to fluctuations in the spiking pattern, activity in one case jumps to *exc:2,1*, and in others it jumps to *exc:2,2*. Also activity in population *exc:3,1* jumps to different destinations.

We have shown in the context of the firing rate model that the network can learn sequences if the interstage feedforward excitatory connections are plastic. For the spiking network, we introduce synaptic plasticity in the feedforward synapses connecting the excitatory populations in one stage to the excitatory populations in the subsequent stage. We use a calcium-based biophysically realistic model of synaptic plasticity (Graupner & Brunel,

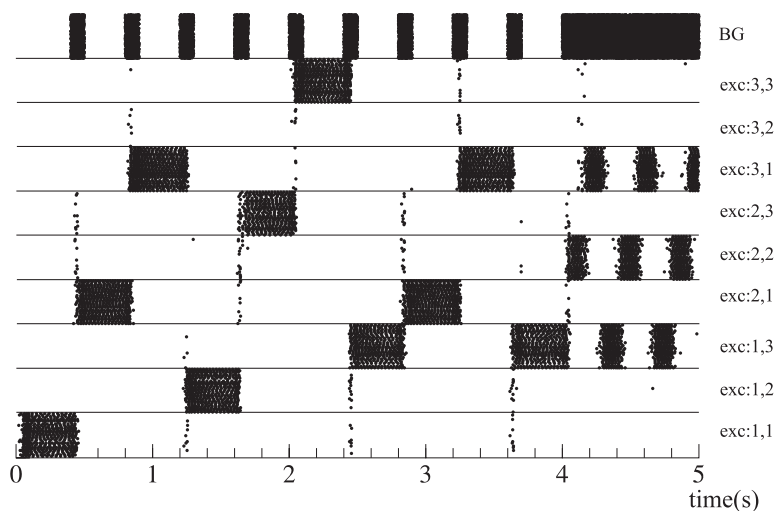


Figure 9: Raster plot of the 30 neurons in each excitatory population in the spiking version of the network in Figure 2. Shown also is the external spike train, BG, used to excite all the excitatory populations. The excitatory-excitatory synapses in the feedforward paths were weakened in order to stop spontaneous propagation of activity. The external spike train, BG, triggers each step in the sequence.

2012) in which presynaptic and postsynaptic spikes trigger a calcium influx due to the activation of *NMDA* receptors and the activation of voltage-dependent calcium channels respectively. The calcium concentration in a synapse modulates its efficacy. Synaptic efficacy has bistable dynamics so that when there is no pre- or postsynaptic activity and the calcium concentration has decayed sufficiently, synaptic efficacy settles to one of two values: high or low. Appendix B contains more details about the synaptic plasticity rule.

Figure 10 shows the simulation results in a longer chain of 10 WTA stages. Each stage has three competing excitatory populations. External excitation that targets one excitatory population in each stage initially sets the path of the sequential activity and reorganizes the feedforward plastic weights to store the input-imposed pattern. External excitation is a 150 Hz Poisson spike train that activates *AMPA*-mediated conductances in the target populations. Between 1 s and 2 s, external excitation steers the sequential activity along a different path that reorganizes the existing pattern of feedforward weights. Sequential activity thus proceeds along the new path after the removal of the input. External excitation also speeds up the propagation of activity as it puts the excitatory neurons closer to threshold and speeds up

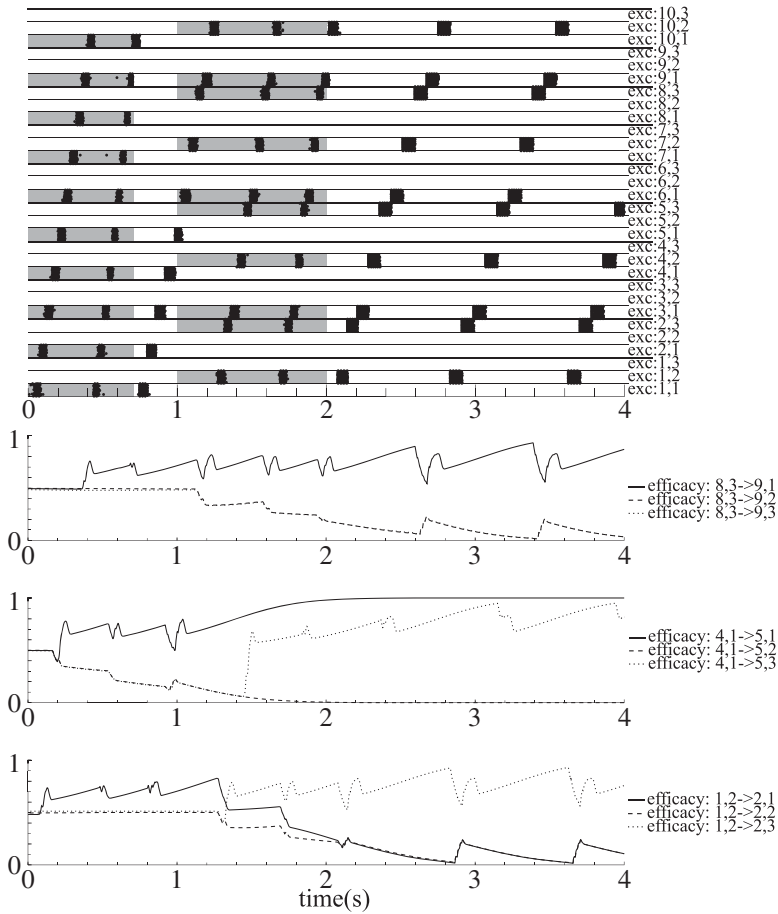


Figure 10: Sequence learning in a spiking network of 10 asymmetrically coupled WTA stages with 3 excitatory populations each and plastic feedforward excitatory-excitatory synapses. Shown are the raster plots of the 30 excitatory populations. A gray rectangle on a population's raster plot indicates external input to that population. The average normalized efficacies of the feedforward plastic connections emanating from three sample excitatory populations (exc:1,2, exc:4,1, and exc:8,3) are shown.

the winner selection process in each stage. Figure 10 also shows a sample of the efficacy of the feedforward connections emanating from some excitatory populations. The efficacy of the feedforward connection between two populations is the average of the efficacy of the 30^2 plastic connections that make up the all-to-all connectivity between the neurons in the two

populations. We ran the simulation in Figure 9 100 times. Each time, the synaptic weights were randomly reinitialized and the Poisson external input recomputed. The network always learns the new input-imposed sequence and reproduces it after the input is removed.

4 Discussion

Networks whose dynamics are governed by fixed-point attractors offer a powerful substrate for implementing a myriad of tasks such as associative memory (Hopfield, 1982), decision making (Wang, 2002, 2008; Soltani & Wang, 2006), and finite state machines (Rutishauser & Douglas, 2009). Stable network states can easily arise if activity in the network stabilizes at a high level due to saturating nonlinearities in the network components. This, however, is not consistent with the general firing pattern of biological neurons, which typically fire at rates far below the maximum rates set by the refractory mechanisms. In order for a network to exhibit nontrivial stable states at realistic activity levels, interacting inhibitory and excitatory neurons are needed (Amit & Brunel, 1997). The asymmetric coupling scheme used to couple WTA circuits in this letter could thus be seen as a general scheme for coupling multiple circuits that individually exhibit stable attractors. The coupling scheme is characterized by feedforward excitatory-excitatory connections from circuit A to circuit B and feedback excitatory-inhibitory connections from circuit B to circuit A, where A and B are arbitrary neural circuits with stable fixed points of activity that arise out of the interplay between excitation and inhibition. The resulting network of coupled circuits will then exhibit well-defined patterns of sequential activity.

One advantage of using a network that is composed of asymmetrically coupled attractor subnetworks in order to generate patterns of sequential activity is noise robustness; the state of each subnetwork is always restored toward a quasi-attractor (Amit, 1989) when the subnetwork is activated. We use the term *quasi-attractor* to denote a point in the state space of the network that exerts a pull on trajectories located in a certain region of the state space. When trajectories from this region approach the quasi-attractor, they are repelled toward another region of the phase space. This acts as a signal restoration mechanism that reduces the network's susceptibility to noise (see Figure 4). Quasi-attractors are not fixed points of the network dynamics. This approach is therefore different from the one that is based on saddle points to guide sequential activity in winnerless competition networks (Rabinovich et al., 2008). In our case, the quasi-attractor point corresponds to the true attractor point that would be present in the uncoupled subnetwork but is not present in the full network due to the asymmetric coupling connections.

If each attractor subnetwork is a cooperative-competitive network (CCN) like the WTA, then there exist multiple competing quasi-attractor states in each of the cooperative-competitive subnetworks that make up the full

network, and multiple patterns of sequential activity can be realized depending on which group of neurons wins the competition in each CCN subnetwork, that is, which quasi-attractor state is approached in each CCN subnetwork. The number of sequences that can arise is exponential in the number of stages or CCN subnetworks cascaded together. This distinguishes our approach from the synfire chainlike models of sequential activity. While synfire chains exhibit a moderate amount of noise robustness (Rotter & Aertsen, 1998), they do not exhibit stable attractors in which sequential activity can temporarily halt, and there is no competition mechanism to enforce a categorical choice between a number of possible sequence paths.

In the proposed network, the activity in one WTA stage can influence the identity of the winning population only in the subsequent stage. Hence, complex sequences in which the identity of one pattern in the sequence is determined by the two or more previous patterns in the sequence cannot be directly implemented. This would require multiple patterns in the sequence to be active simultaneously (Verduzco-Flores et al., 2012), or at least some synaptic trace of their activity to be present, in order to influence the choice of the subsequent pattern in the sequence. As a consequence, the number of distinct sequences that can be stored in the network is limited by the fact that once any two sequences overlap in one position, then they have to be identical in all subsequent positions.

In robotics, robust and configurable neuromorphic generators of sequential activity can enable more human-like movements. A spiking neural network with realistic dynamics (McKinstry & Edelman, 2013) was taught a sequential pattern and was then used to control a robotic arm through a series of movements. The network connectivity was not based on anatomical data. It was not shown whether an external input can choose between a number of possible sequences on the fly and whether the sequence can halt and resume based on the strength of a uniform external input. These last two features are essential if the robotic system is to wait for the consequences of its actions and if it is to respond in real time to its environment. Sequential activity in the network we propose can be triggered and steered in real time. Together with its noise robustness, this makes the proposed network architecture a viable candidate for implementation in neuromorphic hardware that uses either analog or digital neurons (Indiveri et al., 2011).

Dynamics similar to those of WTA networks have been explored in the context of continuous dynamical neural fields (DNF) (Amari, 1977). In DNF, stable peaks of activation are produced and maintained at the point in the field that is receiving the strongest superthreshold input. DNFs have been used in dynamical field theory (DFT) to account for various cognitive functions (Schoener, 2008; Spencer, Perone, & Johnson, 2009). The flexibility of DFT models has been attributed to their ability to go into shallow attractors from which they can easily get out due to the arrival of new input.

In our proposed network, shallow attractors can be obtained by weakening the feedforward excitatory-excitatory connections; a WTA stage can then exhibit a stable activity peak. External input can destabilize this peak. This destabilization does not kill activity in the network but results in a transition where a stable peak in a subsequent WTA stage is created (see Figures 5 and 9). In order to destabilize the stable peak and trigger a transition, the external input has to target the primed WTA stage, that is, the stage that is receiving subthreshold input from the currently active stage. This targeted external input can thus be interpreted as a condition of satisfaction that indicates that the behavioral action associated with the currently active peak has completed and that it is now time to move to the next action (Sandamirskaya & Schöner, 2010). The next action can be determined by either the pattern of plastic feedforward weights or the input if the input targeting the primed WTA stage is stronger for a specific population in that stage. If both mechanisms are in effect and each is trying to pull the network along a different path, then we observe a competition between the external input (which is presumably coming from the environment) and the history of network activity (which has set the pattern of the feedforward plastic connections). Each mechanism is trying to set the path to be followed by the network. Whether the network listens to the input or to the pattern of its internal feedforward weights when choosing the next action is a function of the strength of the input, and the magnitude of the difference between a fully potentiated and a fully depressed synapse. In a spiking network, there can be a considerable element of randomness due to the Poisson external input. A network operating in triggered mode where external input is needed to trigger transitions between shallow attractors could thus serve as the basis for a perception-action loop where the next action is determined by the current active attractor, the external input that is carrying sensory information, and the history of activity in the network. We can have spontaneous transitions interleaved with triggered transitions; the network can thus spontaneously transition through a number of quasi-attractors, where each quasi-attractor is coding for a certain action, and then come to a halt at an attractor and wait for external input to trigger the next action. In summary, the network proposed in this letter can bridge the gap between realistic spiking neural networks with anatomically justified connectivity and the abstract models of cognition coming from dynamical field theory.

5 Conclusion

We presented a cortically inspired recurrent neural network that can generate patterns of sequential activity. This network is noise robust, but its activity pattern can be easily modulated by external input; synaptic plasticity enables the network to learn an input-imposed pattern and reproduce this pattern after the input is removed; the network can exhibit spontaneous

patterns of sequential activity or wait for external input to trigger each transition in the sequence. Each of these individual aspects has been extensively investigated in the literature, but we bring them all together within a unified framework that uses a novel anatomically justified architecture, and biologically realistic neuron, synapses, and spike-based plasticity models. The proposed network elucidates dynamical mechanisms that can potentially underlie sequential activity generation in mammalian cortical circuits. The biologically realistic network elements and network architecture can be used to constrain more phenomenological models of sequential activity and sequential decision making, such as those originating in dynamical field theory. Finally, the noise-robust dynamics of the network make it a good candidate for implementation in neuromorphic hardware devices and application on real-time-behaving robotic platforms.

Appendix A: Analysis of the Rate-Based Model

In this appendix, we provide a simplified analysis of the network shown in Figure 2. We will not investigate the evolution of the network's activity in time, but rather will derive constraints on the population parameters and the connection weights that enable the network shown in Figure 2 to support stable sequential activity. The parameter set that we choose should ensure the elimination of the stable attractors in each individual WTA stage and ensure that the following sequence of events takes place in the correct order in each WTA stage:

1. Activity in the WTA stage ramps up due to excitation from the previous stage until it becomes self-sustaining. A winner is selected.
2. Activity in the winning population reaches a level that enables it to shut down the previous stage through the interstage feedback excitatory-inhibitory connection.
3. Activity in the winning population reaches a level that enables it to activate the subsequent stage and push activity in the winning excitatory population in the subsequent stage beyond the self-sustaining rate.

A.1 Individual WTA Stage. Consider an individual WTA stage made up of a single excitatory population and a single inhibitory population that is described by

$$\tau_e \dot{x}^{exc}(t) + x^{exc}(t) = \text{Max}(0, w^{ee}x^{exc}(t) - w^{ie}x^{inh}(t) - T_e) \quad (\text{A.1})$$

$$\tau_i \dot{x}^{inh}(t) + x^{inh}(t) = \text{Max}(0, w^{ei}x^{exc}(t) - T_i) \quad (\text{A.2})$$

We did not include multiple excitatory populations because we make the simplifying assumption that the winner selection happens so quickly in a

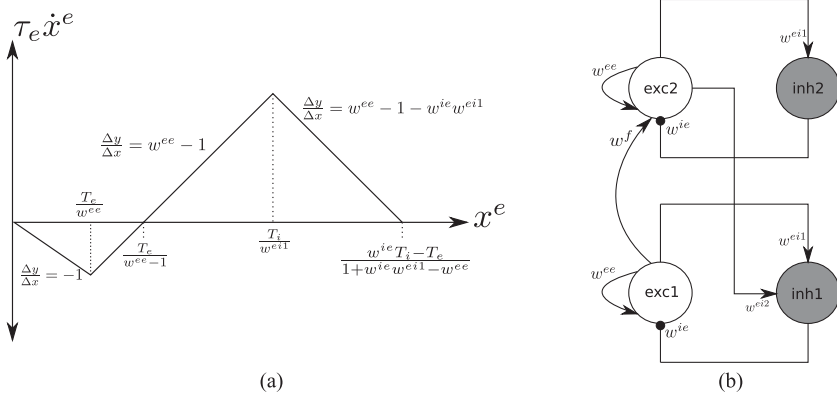


Figure 11: (a) The 1D phase diagram of the system described by equation A.3. (b) Two asymmetrically coupled WTA stages. White circles are excitatory populations, gray circles are inhibitory populations

WTA stage that all but the winning population become quickly suppressed and so only one excitatory population contributes to the dynamics of the WTA stage. We make the additional simplifying assumption that $\tau_i \ll \tau_e$ so that we can reduce the system (given by equations A.1 and A.2) to the 1D system:

$$\tau_e \dot{x}^{exc}(t) + x^{exc}(t) = \text{Max}(0, w^{ee} x^{exc}(t) - w^{ie} \text{Max}(0, w^{ei1} x^{exc}(t) - T_i) - T_e) \quad (\text{A.3})$$

The goal is to find the parameter set that will make this individual, uncoupled stage exhibit a nonzero stable attractor. This can be achieved if the phase space of the 1D system looks like Figure 11a. Each break in the plot corresponds to the second argument of one of the Max functions crossing zero. The necessary constraints are

$$w^{ee} > 1, \quad (\text{A.4})$$

$$w^{ee} - 1 - w^{ie} w^{ei1} < 0, \quad (\text{A.5})$$

$$\frac{T_e}{w^{ee} - 1} < \frac{T_i}{w^{ei1}}. \quad (\text{A.6})$$

The self-sustaining rate is $\frac{T_e}{w^{ee} - 1}$. Beyond that rate, \dot{x}^{exc} is positive, and the excitatory population will approach the steady-state activity in the absence of external input. The steady-state activity is given by

$$x_{SS} = \frac{w^{ie} T_i - T_e}{1 + w^{ie} w^{ei1} - w^{ee}}. \quad (\text{A.7})$$

A.2 Coupled WTA Stages. Consider two WTA stages that are coupled as shown in Figure 11b

$$\tau_e \dot{x}_1^{exc}(t) + x_1^{exc}(t) = \text{Max}(0, w^{ee} x_1^{exc}(t) - w^{ie} x_1^{inh}(t) - T_e), \quad (\text{A.8})$$

$$\tau_i \dot{x}_1^{inh}(t) + x_1^{inh}(t) = \text{Max}(0, w^{ei1} x_1^{exc}(t) + w^{ei2} x_2^{exc}(t) - T_i), \quad (\text{A.9})$$

$$\tau_e \dot{x}_2^{exc}(t) + x_2^{exc}(t) = \text{Max}(0, w^{ee} x_2^{exc}(t) + w^f x_1^{exc}(t) - w^{ie} x_2^{inh}(t) - T_e), \quad (\text{A.10})$$

$$\tau_i \dot{x}_2^{inh}(t) + x_2^{inh}(t) = \text{Max}(0, w^{ei1} x_2^{exc}(t) - T_i). \quad (\text{A.11})$$

Again, we make the simplifying assumption that $\tau_i \ll \tau_e$. Assume that we briefly excite the population x_1^{exc} so that it is approaching the attractor state of the uncoupled stage given by equation A.7. The next stage will be activated when the input to the population x_2^{exc} is above T_e , that is, $w^f x_1^{exc} > T_e$. Hence, using equation A7, we impose the constraint

$$\frac{T_e}{w^f} < \frac{w^{ie} T_i - T_e}{1 + w^{ie} w^{ei1} - w^{ee}}. \quad (\text{A.12})$$

This ensures that the first (bottom) stage activates the second stage before the activity of the first stage settles into the attractor. Activation of the second stage will immediately increase the inhibition in the first stage. The magnitude of the nonzero steady-state activity of x_1^{exc} as a function of x_2^{exc} assuming the latter is kept fixed can be written as

$$x_1^{exc}|_{ss} = \frac{w^{ie}(T_i - w^{ei2} x_2^{exc}) - T_e}{1 + w^{ie} w^{ei1} - w^{ee}}. \quad (\text{A.13})$$

Using this result, we impose a stronger version of condition A.12 to ensure that the excitatory population in the first stage continues to provide superthreshold input to the second stage until the excitatory population in the second stage reaches the self-sustaining rate:

$$\frac{T_e}{w^f} < \frac{w^{ie}(T_i - w^{ei2} \frac{T_e}{w^{ee} - 1}) - T_e}{1 + w^{ie} w^{ei1} - w^{ee}}. \quad (\text{A.14})$$

In deriving the last condition, we have ignored the transients in x_1^{exc} and assumed that it immediately settles into the steady state given by equation A.13 in response to any change in x_2^{exc} . If we keep this assumption, then the increasing activity of x_2^{exc} as it ramps up beyond the self-sustaining rate will at some point drive the activity of x_1^{exc} below the self-sustaining

rate, effectively extinguishing it. We denote the value of x_2^{exc} at which this happens by x^{SD} . Using equation A.13, we can define x^{SD} implicitly:

$$\frac{w^{ie}(T_i - w^{ei2}x^{SD}) - T_e}{1 + w^{ie}w^{ei1} - w^{ee}} = \frac{T_e}{w^{ee} - 1}. \quad (\text{A.15})$$

We impose the additional condition that a WTA stage should shut down the previous stage before activating the next one. Combining this additional condition with condition A.14 and using equation A.15,

$$x^{SD} = \frac{T_i(w^{ee} - 1) - T_e w^{ei1}}{w^{ei2}(w^{ee} - 1)} < \frac{T_e}{w^f} < \frac{w^{ie}(T_i - w^{ei2}\frac{T_e}{w^{ee}-1}) - T_e}{1 + w^{ie}w^{ei1} - w^{ee}}. \quad (\text{A.16})$$

We found that by satisfying the conditions A.4–A.6 and A.16, we could obtain well-defined sequential activity. Complications may arise when there are multiple excitatory populations in a WTA stage that receive very similar inputs from the previous stage. In that case, our assumption that only one excitatory population contributes to the dynamics of a WTA stage fails. Multiple active excitatory populations in a single stage may cooperate to shut down the previous stage before any of them has reached the self-sustaining rate, and then they all decay to zero in the absence of input. We could easily avoid this problem, however, by choosing a smaller value for w^{ei2} , the feedback excitatory-inhibitory connection weight, that continues to satisfy the left inequality in condition A.16. The parameter set that we used in the simulations of the rate-based model is given in Table 1. *Uniform*(min, max) indicates a uniform distribution on the interval [min, max].

Appendix B: Details of the Spiking Model

The models and the parameter values used in the spiking network closely follow those presented in Wang (2002). The three types of synaptic currents in the neuron equation, equation 3.1, are modeled as

$$I_{AMPA}(t) = (V_E - V_m(t)) \sum_i w_{AMPA}^i s_{AMPA}^i(t) \quad (\text{B.1})$$

$$I_{NMDA}(t) = \frac{(V_E - V_m(t))}{1 + \exp(-62 * V_m(t))/3.57)} \sum_i w_{NMDA}^i s_{NMDA}^i(t) \quad (\text{B.2})$$

$$I_{GABA}(t) = (V_I - V_m(t)) \sum_i w_{GABA}^i s_{GABA}^i(t). \quad (\text{B.3})$$

V_E and V_I are the reversal potentials for the excitatory and inhibitory synapses, respectively. w_{AMPA}^i , w_{NMDA}^i , and w_{GABA}^i control the magnitude

Table 1: Parameter Values for the Rate-Based Model.

Parameter values for the network shown in Figure 2 and described by equation 2.1	
w^{ee}	1.9
w^{ei1}	0.7
w^{ei2}	0.3
w^{ie}	1.5
$w^{lateral}$	0.3
T_e	4
T_i	9
τ_e	0.04
τ_i	0.01
$w_{i,j,k}^f$ used in Figures 3, 4, 6	Independently sampled from Uniform[0.095,0.105]
$w_{i,j,k}^f$ used in Figure 5	Independently sampled from Uniform[0.045,0.055]
Parameter values for the plastic connections described by equation 2.3	
w_{min}	0.08
w_{max}	0.12
v_{th}	8
τ_w	1
K	0.01

of the changes in synaptic conductances in the the synapses formed by presynaptic neuron i when neuron i spikes (the first two are zero if i is an inhibitory neuron; the third is zero if i is an excitatory neuron). Equation B.2 models the voltage-dependent magnesium blockage of the NMDA receptor-activated ion channel (Jahr & Stevens, 1990). The extracellular magnesium concentration is assumed to be 1 mM. $s_{AMPA}^i(t)$, $s_{NMDA}^i(t)$, and $s_{GABA}^i(t)$ reflect the time course of the change in synaptic conductances in response to spikes from neuron i :

$$\dot{s}_{AMPA}^i(t) = -\frac{s_{AMPA}^i(t)}{\tau_{AMPA}} + \sum_k \delta(t - t_k^i) \quad (B.4)$$

$$\dot{s}_{NMDA}^i(t) = -\frac{s_{NMDA}^i(t)}{\tau_{NMDA}^{fall}} + \frac{x^i(t)}{\tau_{NMDA}^{rise}} (1 - s_{NMDA}^i(t)) \quad (B.5a)$$

$$\dot{x}(t) = -\frac{x(t)}{\tau_{NMDA}^{rise}} + \sum_k \delta(t - t_k^i) \quad (B.5b)$$

$$\dot{s}_{GABA}^i(t) = -\frac{s_{GABA}^i(t)}{\tau_{GABA}} + \sum_k \delta(t - t_k^i) \quad (B.6)$$

where t_k^i is the k th spike emitted by neuron i . The τ_{AMPA} , τ_{NMDA}^{fall} , τ_{NMDA}^{rise} and τ_{GABA} time constants control the time course of conductance changes. The rise times of the GABA and AMPA-mediated conductances are neglected.

The membrane capacitance, leak conductance, and refractory period of an excitatory neuron are denoted by C_m^{exc} , g_l^{exc} , and T_{ref}^{exc} , respectively, and those of an inhibitory neuron by C_m^{inh} , g_l^{inh} , and T_{ref}^{inh} . Excitatory and inhibitory neurons share the same firing threshold, V_{firing} , and resting and reset potential, V_i . Each excitatory population and each inhibitory population in Figure 2 is made up of 30 neurons. Each neuron is described by equation 3.1. An arrow in Figure 2 represents all-to-all connectivity from neurons in the source population to neurons in the target population. All connections originating from excitatory neurons give rise to excitatory postsynaptic currents mediated by both *NMDA* and *AMPA* receptors. All connections originating from inhibitory neurons give rise to inhibitory postsynaptic currents mediated by *GABA_A* receptors. The magnitude of the changes in the synaptic conductances due to a presynaptic spike (the w terms in equations B.1, B.2, and B.3) is drawn from random distributions with mean w_{AMPA}^{ee} and w_{NMDA}^{ee} for the recurrent excitatory synapses, w_{GABA}^{ie} for the inhibitory-excitatory synapses, w_{AMPA}^{ei1} and w_{NMDA}^{ei1} for the intrastage excitatory-inhibitory synapses, w_{AMPA}^{ei2} and w_{NMDA}^{ei2} for the interstage feed-back excitatory-inhibitory synapses, w_{AMPA}^f and w_{NMDA}^f for the interstage feedforward excitatory-excitatory synapses, and w_{AMPA}^{lat} and w_{NMDA}^{lat} for the lateral excitatory-excitatory synapses between neurons in different populations within each stage. The magnitudes of conductance changes (the weights) are chosen independently for each synapse at the beginning of the simulation and kept fixed during the simulation. External excitation used to initiate, trigger, or steer the sequential activity takes the form of a Poisson spike train that activates *AMPA* receptor-mediated conductances in neurons in the target population. The magnitude of the jump in the *AMPA* conductance of each neuron in the target population due to an external spike is drawn from a gaussian distribution with mean $w_{AMPA}^{external}$. The parameters of the spiking model are given in Table 2.

For the simulation in Figure 10, we used the synaptic plasticity model proposed by Graupner and Brunel (2012) to modulate the magnitudes of the feedforward *NMDA* receptor-mediated conductances, w_{NMDA}^f . For convenience, we reproduce the model below. Synaptic plasticity is governed by the calcium concentration $C(t)$ in the synapse

$$\dot{C}(t) = \frac{-C(t)}{\tau_{CA}} + C_{pre} \sum_k \delta(t - t_k^{pre}) + C_{post} \sum_k \delta(t - t_k^{post}). \quad (B.7)$$

τ_{CA} is the time constant of calcium concentration decay, t_k^{pre} and t_k^{post} are the times of the k th presynaptic spike and k th postsynaptic spike, respectively, and C_{pre} and C_{post} are the jumps in calcium concentration due to presynaptic spikes and postsynaptic spikes, respectively. The synaptic efficacy, $\rho(t)$,

Table 2: Parameter Values for the Spiking Network.

Parameter	Value	Parameter	Mean Value	Mean/SD
C_m^{exc}	0.5 nF	$w_{AMPA}^{external}$ (gaussian)	$4.0 * 10^{-9}$	2.5
C_m^{inh}	0.2 nF	w_{AMPA}^{ee} (gaussian)	$1.0 * 10^{-10}$	2.5
g_l^{exc}	25 nS	w_{NMDA}^{ee} (gaussian)	$8.0 * 10^9$	2.5
g_l^{inh}	20 nS	w_{GABA}^{ie} (gaussian)	$2.0 * 10^{-9}$	2.5
T_{ref}^{exc}	2 ms	w_{AMPA}^{ei1} (gaussian)	$1.0 * 10^{-9}$	2.5
T_{ref}^{inh}	1 ms	w_{NMDA}^{ei1} (gaussian)	$4.0 * 10^{-9}$	2.5
V_{firing}	-50 mV	w_{AMPA}^{ei2} (gaussian)	$3.0 * 10^{-9}$	2.5
V_L	-70 mV	w_{NMDA}^{ei2} (gaussian)	$7.0 * 10^{-9}$	2.5
V_E	0 V	w_{AMPA}^f (gaussian)	$1.0 * 10^{-10}$	2.5
V_I	-70 mV	w_{NMDA}^f (gaussian) in Figure 8	$2.9 * 10^{-9}$	2.5
τ_{AMPA}	2 ms	w_{NMDA}^f (gaussian) in Figure 9	$2.0 * 10^{-9}$	2.5
τ_{NMDA}^{fall}	100 ms	w_{AMPA}^{lat} (Gaussian)	$1.0 * 10^{-11}$	2.5
τ_{NMDA}^{rise}	2 ms	w_{NMDA}^{lat} (Gaussian)	$1.0 * 10^{-10}$	2.5
τ_{GABA}	5 ms			

varies in the range $[0, 1]$ according to

$$\begin{aligned} \tau_\rho \dot{\rho}(t) = & -\rho(t)(1 - \rho(t))(0.5 - \rho(t)) + \gamma_p(1 - \rho(t))\Theta(C(t) - \theta_p) \\ & - \gamma_d \rho(t)\Theta(C(t) - \theta_d). \end{aligned} \quad (B.8)$$

τ_ρ is the time constant of efficacy changes. $\Theta(x)$ is the Heaviside step function, γ_p and γ_d are the potentiation and depression rates, respectively, and θ_p and θ_d are the calcium concentration thresholds for inducing potentiation and depression, respectively. In the absence of pre- and postsynaptic activity, the efficacy, $\rho(t)$, drifts to one of the two fixed points: 0 or 1. We use $\rho(t)$ to modulate the magnitude of each *NMDA*-mediated conductance in the feedforward path (from a neuron in an excitatory population in one stage to a neuron in an excitatory population in the subsequent stage). The conductance magnitude is modulated between two values, w_{\min} and w_{\max} :

$$w_{NMDA}^f(t) = \rho(t)(w_{\max} - w_{\min}) + w_{\min} \quad (B.9)$$

The parameter values for the plasticity model are given in Table 3.

Table 3: Parameter Values for the Plasticity Model in the Spiking Network.

τ_ρ	100 ms
τ_{CA}	20 ms
C_{pre}	1
C_{post}	2
γ_p	5
γ_d	1
θ_p	1.5
θ_d	0.5
w_{min}	$3 * 10^{-9}$
w_{max}	$3.4 * 10^{-9}$

Notes: The NMDA conductance plasticity model is described in equations B.7 to B.9. This plasticity model is used in the simulations shown in Figure 10.

Acknowledgments

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